"A STUDY OF POSTERIOR CIRCULATION ISCHEMIC STROKE IN 80 PATIENTS – CLINICAL MANIFESTATIONS, RISK FACTORS, IMAGING STUDIES AND OUTCOME"

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CERTIFICATE

This is to certify that this dissertation work entitled **"A STUDY OF POSTERIOR CIRCULATION ISCHEMIC STROKE IN 80 PATIENTS – CLINICAL MANIFESTATIONS, RISK FACTORS, IMAGING STUDIES AND OUTCOME"** is a bonafide record of work done by **Dr.R.ARIVALAGAN**, in the **DEPARTMENT OF MEDICINE**, P.S.G. Institute of Medical Sciences & Research, Coimbatore-641 004, under my supervision and guidance.

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CONTENTS

INTRODUCTION

Stroke is one of the leading causes of morbidity and ranks next only to coronary artery disease and malignancy as the leading cause of mortality worldwide. At least fifty percent of neurological disorders in a general hospital are due to stroke.

As remarked by a renowned neurologist, C. M. Fisher, neurology is learnt stroke by stroke.

Eighty percent of stroke is ischemic; twenty percent of ischemic events involve tissue supplied by posterior circulation. The paralysis of vertebrobasilar stroke can devastating and some forms have high rates of death. Many cases of vertebrobasilar diseases remain undiagnosed or are incorrectly diagnosed. Formerly clinicians used the catchall term vertebrobasilar insufficiency to indicate a hemodynamic cause of all cases of posterior circulation ischemia.

During the past fifteen years information provided by detailed clinical studies and brain imaging has revolutionized our understanding of clinical aspects, mechanism, treatment and prognosis of posterior circulation ischemic stroke.

The etiology of posterior circulation ischemia has been thought to be primarily due to local arterial atherosclerosis (large artery disease) and penetrating artery disease (lacunes) However there is increasing evidence

that cardiogenic embolization is more common than previously suspected and is responsible for 20 – 50% of posterior circulation strokes.

The posterior circulation, unlike the intracranial portions of the anterior circulation, is prone to atherosclerosis much as other systemic arteries. In the case of one vertebral artery being occluded, collateral flow comes from the opposite vertebral artery, from muscular cervical artery branches, and from posterior communicating artery.

Now with better understanding of risk factors involved in stroke the emphasis should be prevention rather than management.

AIMS OF THE STUDY

To evaluate the etiology in posterior circulation stroke.

To study the commonest mode of clinical presentation in posterior circulation ischemic stroke.

To evaluate the commonest anatomical area involved and the size of the infract with the help of imaging modalities (CT and MRI).

To assess the outcome.

REVIEW OF LITERATURE

Definition

According to WHO stroke is defined as rapidly developing clinical signs of local or global disturbance of cerebral function lasting more than 24hrs or leading to death with no apparent cause other than vascular

The term transient ischemic attack implies a complete recovery of cerebral function within 24hrs. Other sub types of stroke include cerebral hem**o**rrhage, sub arachnoids hemorrage, cortical venous sinuses thrombosis.

The normal brain is dependent upon a relatively constant supply of oxygen, glucose and other nutrients derived from the blood perfusing it. Normal blood flow to brain is 55to 75ml per minute. If for any reason blood flow is reduced below 15ml per 100gm per minute, the resulting ischemia with hypoxia when sufficiently prolonged may result in death of neurons and glia [cerebral infarction]

The mean arterial blood pressure, cerebral vascular and tissue resistance, local metabolic products together with several known and unknown factors help to maintain the critical threshold of blood flow for energy metabolism. Further more the blood varies in different areas of brain and autoregulation determines the regional blood flow to meet the local metabolic needs.

In regions of cerebral ischemia there is paralysis of auto regulations and the microvasculature here is not reactive to pressure changes and vasoactive agents and to other forms of stimuli. The cerebral vasculature in this ischemic zone becomes permeable to protein and fluid leaks in the vicinity leading to extra cellular edema. Such events also lead to hemoconcetration and vascular stasis. Hence cerebral infarction is not merely a result of ischemia from occluded vessels but an end result of a series of highly complex ischemia modifying agents.

EPIDEMIOLOGY OF STROKE

Strokes are not only less frequent than coronary events but they occur later in life, and so in many cohort studies even with prolonged follow-ups, the number of incident strokes is still relatively small.

The diagnosis of stroke is still a matter of clinical skill. It is a disorder of late middle age and the elderly, where other diseases frequently coexist to confuse the situation. Also stroke are pathologically more diverse from coronary events, which are mostly due to thrombotic and embolic complication of atheroma. On the other hand stroke may be due to intracranial small vessel disease which is different from atheroma, embolism from heart, primary intracerebral hemorrhage and subarachnoid hemorrhage. Therefore the incidence and etiology of stroke should be considered separately but in many large epidemiology studies this has been impossible due to lack of imaging modalities and also due to inability to ascribe precise cause for stroke in many patients.

Stroke mortality is high in Eastern Europe and Japan. In North America and some Western European countries the rates are low. A reason for higher mortality in Japan and Eastern Europe may be due to the number of increased cases which occur there, however no data is available for the same.

INCIDENCE

Incidence in UK is 2per 1000 per year. About 100, 000 patients have first stroke every year, one every 5 minutes. Most of the cases occur above 65yrs. 25% occurs before 65yrs. Apart from SAH, it is impossible to separate strokes reliably during life into pathological types without CT scanning so until recently no studies could give the correct incidence.

PREVALENCE

Typical estimate of stroke prevalence Is 5/1000 population but clearly exact number depends on population and sex structure and becomes 50/1000 in men and 25/1000 in women between age groups of 65-74. Prevelance is important for planning purposes the prevalence of disability in general is far more important than just stroke related disability, particularly when one comes to consider elderly populations who have so many additional causes of disability impossible to disextemple from stroke related disability. Such as arthritis, claudtication, and dementia. prevelance is difficult to measure because of the large numbers involved and due to the non reliable history there is always chances of diagnostic error in retrospective surveys. Also stroke prevalence depends on incidences of incidences of survival both of which may vary by time and place.

Racial and social influences also play major role in stroke. As for as the mortality rate is concerned it is rapidly declining compared to coronary artery disease in most places except in Eastern Europe. If the incidence is really declining the reasons are unclear, but there must be environmental and so potentially modifiable, rather than genetic causes like rheumatic heart diseases are less than it was, but accurate data is hard to come by. Seasonal and diurnal variation plays a part, as the admissions for stroke are higher in winter than in summer.

HISTORICAL PERSPECTIVES

One of the first person who evinced keen interest in cerebrovascular system was SIR THOMAS WILLIS who coined the term NEUROLOGY. He performed necropsies on his patients and did extensive anatomical dissection on the brain. After him there was a lull until the later years of 19th century when physicians, mostly in France, Germany and UK who helped to elucidate the anatomy and functioning of Brainstem[1], most of the brainstem lesions syndrome were found out during this period. The next major contribution for the enhancement of neurology was Joseph Jules Djerrine and his wife Augustine Klumpkes. Dejjerine described the findings in patients with different varieties of reading abnormalities and first described the syndrome of alexia without agraphia[2].

Charls Foix was probably the first modern stroke neurologist[3, 4]. In a span of 3years he and his colleague published reports on the clinico anatomical correlation of symptoms and signs with softenings at various sites in cerebral hemispheres in the brainstem[5], especially important in the

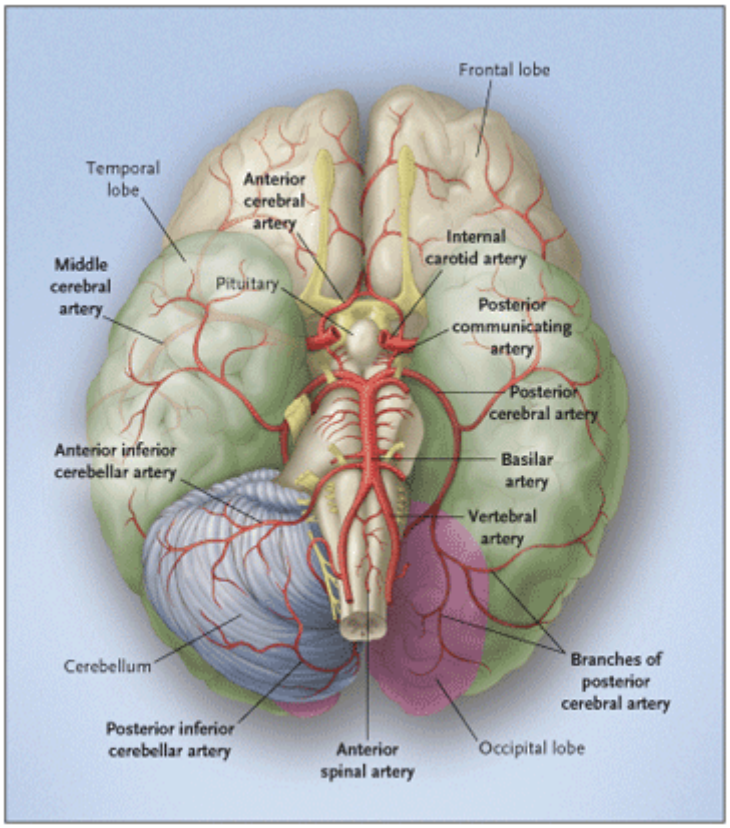
posterior circulation where his studies on THALAMIC syndrome related to occlusion of posterior cerebral arteries and LATERAL MEDULLARY syndrome.

Later clinicians clarified the clinical findings in patients with pontine infarction related to various loci in the cerebellum, midbrain[6], thalamus and occipital and temporal lobe infarction in patients with embolism to the "top of basilar" artery[7], and patients with small localized infarcts in the pons medulla, thalamus caused by disease of the penetrating artery supply[8-15].

POSTERIOR CIRCULATION BLOOD SUPPLY

The posterior circulation is constructed quite differently from the anterior circulation and consists of vessels from each side which unite to form Midline arteries that supply the brainstem and spinal cord within the posterior circulation, there is a much, higher incidence of asymmetric, hypoplastic arteries, of variability of supply and of retention of fetal circulatory patterns.

The proximal portions of the posterior circulation on the two sides differ on the right the subclavian artery arises from the in nominate artery, a common channel supplying the anterior and posterior circulation. On the left side, the subclavian artery usually arises directly from the aortic arch after the origin of the left common carotid artery.



The first branch of each subclavian artery is the vertebral artery (VA). The Vertebral arteries[16] course upward and backward until they enter the transverse foramens of C_6 or C_S and run within the intravertebral foramen exiting to course behind the atlas before piercing the duramater to enter the foramen magnum. Their intracranial portions end at the medullopontine junction, where the two vertebral arteries join to form the basilar artery.

The first portion of Vertebral artery before entry into the bony vertebral column is (V_1), the portion within the vertebral columns (V_2), and the portion after exit form the vertebral column that arches behind the atlas

and before entry into the cranium (V_3) and the intracranial portion is V_4. In the neck, vertebral arteries have many small muscular and spinal branches.

The intracranial portion of the vertebral arteries gives off posterior and anterior spinal artery branches penetrating arteries to the medulla and the large posterior inferior cerebellar arteries (PICAs)

The posterior inferior cerebellar artery (PICA) in its proximal segment supplies the lateral medulla and in its distal branches, the inferior surface of the cerebellum. Anastomotic channels exist among the ascending cervical arteries, the thyro cervical arteries, the occipital artery and the second segment of the vertebral artery.

The basilar artery runs in the midline along the clivus, giving off bilateral anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA) branches before dividing at the pontomesencephalic junction into terminal Posterior cerebral artery branches.

Large paramedian arteries and smaller short circumferential arteries penetrate through the basal portion of the brain stem in to the tegmentum. Long circumferential arteries course around the brainstem giving off braches to the lateral tegmentum.

In 75% of cases, both posterior cerebral arteries arises from the bifurcation of the basilar artery, in 20% one has its origin form the ipsilateral internal carotid artery via the posterior communicating artery, in 5% of cases, both originate from the respective, ipsilateral internal carotid arteries. The precommunal, or P_1 segment of the true posterior cerebral artery is atretic in such cases.

The P_1 segment of the true posterior cerebral gives rise to small brainstem Penetrating branches that supply the middle cerebral peduncles, the substantia formation, subthalamic nucleus, decussation of the superior cerebellar peduncles, the medial longitudinal fasciculus and the medial lemniscus

The artery of percheron arises from either the right or the left precommunal segment of the posterior cerebral arteries it divides in the subthalamus to supply the inferomedial and anterior portions if the thalamus, and subthalamus bilaterally. The thalamogeniculate branches, and subthalamus bilaterally. The thalamogeniculate branches, which also orginate from the precommunal portion of the posterior cerebral artery supply the dorsal, dorsomedial, anterior and inferior thalamus, and the medial geniculate body.

The medial posterior choroidal artery supplies the superior dorsomedial and dorsoanterior thalamus and the medial geniculate body in addition to the tela choroidea of the third choroid plexus of the lateral ventricle.

The P_2 segment of the posterior cerebral arteries gives off small circumferential branches that course around the midbrain to supply the lateral part of cerebral peduncles, medial lemniscus tegmentum of the midbrain, superior calliculi, lateral geniculate body, posterolateral nucleus of the thalamus, choriod plexus and hippocampus.

The cortical branches of posterior cerebral artery includes Anterior temporal artery, posterior temporal artery, calcaine artery and parieto occipital artery which supplies inferomedial temporal lobe, parahippocampal

and hippocampal gyri and occipital cortex including the primary visual cortex and the visual association areas.

The Posterior cerebral arteries give off penetrating arteries to the midbrain and thalamus and course around the cerebral peduncle and then supply the occipital lobes and inferior surface of temporal lobes

PHYSIOLOGY OF BLOOD FLOW IN BRAIN

The brain is a metabolically active organ. The brain uses glucose as its sole substrate for energy metabolism. A constant supply of Adenosine tri phosthate (ATP) (Derived from glucose metabolism) is needed to maintain neuronal integrity and to keep the major extra-cellular cations Ca^{++} and NA^{++} outside the cells and the intracellular cation K^{++} within the cells.

The brain requires and uses approximately 500 CC of oxygen and 75 – 100 mg of glucose each minute and a total of 125g of glucose each day[17]

The brain uses approximately 20% of cardiac output when the body is resting [17]. Cerebral blood flow is minute and cerebral 50ml for 100g of brain tissue per minute and cerebral oxygen consumption is normally approximately 3. 5CC / 100g / minute.

Brain energy use and blood flow depend on the degree of neuronal activity. PET and functional magnetic resonance imaging show that using the right hand increases, metabolism and cerebral blood flow in left motor cortex.

The capacity of the cerebral circulation to maintain relatively constant level of cerebral blood flow despite changing blood pressure, has traditionally been termed auto regulation. Cerebral blood flow remains constant when mean arterial blood pressures are between 50 and 150mm of Hg

FACTORS AFFECTING TISSUE SURVIVAL

The survival of the brain regions at risk depends on a number of factors[18]:

(1) The adequacy of collateral circulation
(2) The state of the systemic circulation
(3) Serologic factors
(4) Change within the obstruction vascular lesion, and
(5) Resistance within the microcirculatory bed.

Events during first three weeks after vascular occlusion:

Experience shows that the tenuous balance created by occlusion of a major artery is temporary and usually resolves in 2-3 weeks at most. During this period, any systemic changes, such as decrease in fluid volume or positional or pharmacologically mediated drops in blood pressure, can cause worsening of symptoms. By three weeks either the brain tissue has died causing brain infarct, or collateral sources of blood flow develop which adequately supply the region at risk. By 2-3 weeks collateral circulation stabilizes and the patients are vulnerable to positional or circulatory changes. By 2-3 weeks the clots has become more adherent and has much less tendency to embolise. Most studies of patients with anterior and

posterior circulation ischemia document a low frequency of progression of acute ischemic events after 2 weeks.

During the hours, days and early weeks after a vascular occlusion, the question of death or survival of at-risk brain tissue can be viewed as a clash between factors acting to worsen ischemia and natural body responses that prevent or limit ischemia. Clinicians hope to build on the body's natural defenses and counteract the factors that promote ischemia.

This process of shifting vulnerability translates clinically into fluctuating variables symptoms and signs during the early period after a vascular occlusion. Acute blockade of artery often translates into the sudden onset of symptoms. After vascular occlusion, a weighing of the balance of positive and adverse factors towards the adverse side causes transient deficits or neurologic symptoms and signs. Sudden worsening often related to distant embolization.

Pathology Of Ischemic Mechanisms Of Cerebrovascular Damage To Brain Tissue

Ishemia can be further subdivided into three different mechanisms:
Thrombosis,
Embolism, and
Decreased systemic perfusion.

Thrombosis –VIRCHOW[19] introduced the terms thrombus, thrombosis, embolus and embolism and deduced the general principles of thrombosis and embolism, virchows triad explained localized thrombus formation and consisted of the following

1. An abnormality of the intima and vascular wall
2. An abnormality of blood flow, and
3. An abnormality of blood coagulability.

By convention thrombosis refers to an obstruction of blood flow to a localized occlusive process within one or more blood vessels. The lumen of the vessel is narrowed by an alteration in the vessel wall or by superimposed clot formation. The most common type of vascular pathology is atherosclerosis. Atherosclerosis affects chiefly the larger extra cranial and intracranial vessels[20, 21]. Occasionally, a clot forms within the lumen because of a primary hematologic problem, such as polycythemia, thrombocytosis, or a systemic hyper coagulability. The smaller penetrating intracranial vessels are more often damaged by hypertension than by atherosclerotic processes. In such cases increased arterial tension leads to hypertrophy of the media and deposition of fibrinoid material into the vessel wall, a process that gradually encroaches on the already small lumen. Atheromatous plaques often referred to as microatheromas, can obstruct the orifices of penetrating arteries

Less common vascular pathologies leading to obstruction include

1. Fibromuscular dysplasia, an overgrowth of medial and intimal elements that compromise vessel contractilityand lumina size
2. Arteritis especially of the takayasu or giant cell type
3. Dissection of the vessel wall
4. Hemorrhage into plaque.

At times the focal vascular abnormality is a functional change in the contractility of blood vessel. Intense focal vasoconstriction can lead to decreased blood flow and thrombosis. Dilatation of blood vessels also alters local blood flow and clots often form in dilated segments.

EMBOLISM

In embolism material formed elsewhere within the vascular system lodges in a vessel and blocks the blood flow. In contrast to thrombosis, embolic luminal blockage is not caused by a localized process originating within the blocked vessel. The material arises proximally, most commonly from the heart, from major arteries such as aorta, carotids, and vertebral arteries and from systemic veins. Cardiac sources s of embolism includes the heart valves endocardium and clots or tumors within the atrial or ventrical cavities. Artery to artery emboli are composed of clot platelets clumps or fragments of plaques that breakoff from proximal vessels. Clots originating in systemic veins travel to the brain through cardiac defects such as atrial septal defect or a patent foramen ovale a process termed paradoxical embolism[22]. Also currently air, fat, plaque material particulate

matter from injected drugs, bacteria and tumor cells enter the vascular system and embolise to brain vessel.

DECREASED SYSTEMIC PERFUSION

In decreased systemic perfusion, diminished flow to brain tissue is caused by low systemic perfusion pressure. The most common causes are cardiac pump failure and systemic hypotension. In such cases, the lack of perfusion is more generalized than in localized thrombosis or embolism and affects the brain diffusely and bilaterally. Poor perfusion is most critical in border zone or watershed regions at the periphery of the major vascular supply territories

DAMAGE CAUSED BY ISCHEMIA

All three mechanisms of ischemia lead to temporary or permanent tissue injury. Permanent injury is termed infarction. Capillaries or other vessels within the ischemic tissue may lead to leakage of blood into the ischemic tissue resulting in hemorrhagic infarcts. The extent of brain damage depends on the location and duration of the poor perfusion and the ability of the collateral vessels to perfuse the tissue at risk. Brain and vascular injuries may lead during the hours and days after stroke to brain edema. In the chronic phase, glial scars forms and macrophages gradually ingests the necrotic debris within the infarct, leading to shrinkage of the volume of the infracted tissue or to formation of a frank cavity.

Distribution of Vascular pathology Thrombosis:

Sites of predilection for atherosclerotic narrowing in the posterior circulation include the proximal origins of vertebral arteries and the subclavian arteries, the proximal and distal ends of intracranial vertebral arteries, the basilar artery, and the origin of the Posterior cerebral arteries [23]. Atherosclerotic narrowing rarely affects the distal superficial branches like Posterior inferior cerebellar artery, anterior inferior cerebellar artery and Superior carebellar arteries.

Lipohyalinosis and medial hypertrophy secondary to hypertension affect mainly the Thalamo geniculate penetraters from the Posterior cerebral arteries and paramedian perforating vessels to the pons, midbrain and thalamus from the basilar artery[24].

Atheroma formation or emboli that lodge at the top of the basilar artery or along the P_1 segment may cause symptoms by occluding one or more of the small brainstem – penetrating branches. Occlusion in the posterior cerebral artery distal to the junction with the posterior communicating artery (P_2) may disrupt small circumferential branches.

Atherothrombotic lesions have predilections for V_1 and V_4 segments of the vertebral artery. The first segment may become diseased at the origin of the vessel and may produce posterior circulation emboli. Collateral flow from the contralateral vertebral arteries is usually sufficient to prevent low flow Transient ischemic attacks or stroke.

When one vertebral artery is atretic and an atherothrombotic lesion threatens the origin of the other, the collateral circulations, which may also include retrograde flow down the basilar artery, is often insufficient. In this setting, low flow Transient ischemic attacks may occur, consisting of syncope, vertigo, and alternating hemiplegia. Disease of the distal fourth segment (V_4) of the vertebral artery can promote thrombus formation manifest as embolism or with propagation of basilar artery thrombosis. Stenosis proximal to the origin of the posterior inferior cerebellar artery can threaten the lateral medulla and the posterior inferior surface of the cerebellum.

If the subclavian artery is occluded proximal to the origin of the vertebral artery, there is reversal in the direction of blood flow in the ipsilateral vertebral artery. Exercise of the ipsilateral arm may increase demand on vertebral flow, producing posterior circulation TIA's 'subclavian steal syndrome'.

Although atheromatous disease rarely narrows the V_2 and V_3 segments of the vertebral artery, this region is subject to dissection, fibromuscular dysplasia and rarely encroachment by osteophytic spurs within the vertebral foramina.

Atheromatous lesions can occur anywhere along the basilar trunk but are most frequent in the proximal basilar and distal vertebral segments.

Embolism from heart or proximal vertebral or basilar segments are more commonly responsible for 'Top of the basilar' syndromes.

Dissection, traumatic or spontaneous usually involves the distal extra cranial carotid and vertebral arteries. Temporal arteries affects the vertebral arteries just before they pierce the dura to enter the cranial cavity[25].

Emboli can block any artery depending on the size and the nature of the embolic materials. In posterior circulation, emboli preferably block the intracranial vertebral artery, distal basilar artery and the Posterior cerebral arteries.

The extent and size of the infarct depend on the rate of occlusion, adequacy of collateral circulation, and resistance of brain structures to lschemia. The distribution of damage in patients with low flow is the concept of distal field's ischemia.

Risk Factors

Risk factors are characteristics of an individual or of a population. Risk factors for posterior circulation stroke are the same as for other forms of cardiovascular disease. These are either modifiable or non-modifiable. The non-modifiable risk factors include age, gender, race, family history of stroke or TIA. The modifiable risk factors include hypertension, diabetes mellitus, heart disease, such as atrial fibrillation, ischemic heart disease, rheumatic heart disease, hyper coagulabe states, antiphospholipid antibody syndrome, homocystinemia, etc. ,

Age

Strongest risk factor for ischemic stroke, intracerebral haemmorage and sub arachnoid haemmorhage and certainly for sub types of ischemic stroke as well. stroke in people aged 75-84[26] is about 25 times more common than in aged 45-54.

Sex

There is a small exces in male, which is more common in middle age to old age, disappearing in the elderly and probably absent in the young. Even though most stroke are due to infarction and most infarcts are due to atheroembolism.

BLOOD PRESSURE

Blood pressure is strongly associated with stroke. most information comes from consideration of diastolic pressure, the relationship with systolic pressure is similar and possibly stronger and even isolated systolic pressure is associated with increased risk. , because the strength of blood pressure and stroke association is so strong, consistent and biologically plausible and because treatment of hypertension reduces stroke risk, one can be sure that hypertension is a causal risk factor, atleast for stroke in general. Because hypertension is prevelant in population at large, it is greater than any other risk factor overall association between hypertension and stroke is less in the elderly than in middle aged, hypertension increases the stroke risk by increasing the extent and severity of atheroma and the prevalence of small vessel disease in perforating arteries within the brain.

CIGARETTE SMOKING

It is a relative risk of about 2% for ischemic stroke. Smoking has been related to the extent of carotid disease in patients selected for angiography, by ultrasound and in identical twins discordant for smoking.

LIPIDS

The relationship between blood lipids and stroke is not as strong as its relationship with coronary artery disease lipprotien [a] is perhaps predictive. Some attempts to relate atheroma in the extra and intracranial circulation to blood lipids concentration has suggested an association. So rather surprisingly therefore cholesterol lowering drugs seem to reduce stroke risk, albeit in fairly low risk populations.

DIABETES MELLITUS

Diabetes mellitus doubles the risk of stroke compared to non-diabetics, probably independent of any association with other risk factors such as hypertension. However care must betaken when interpreting any relationship between diabetics and mortality, since stroke in diabetes are more likely to be fatal.

HAEMOSTATIC VARIABLES

Increased fibrinogen levels lead to an accelerated thrombus formation raised plasma factor vii coagulant activity, raised tissue plasminogen

activator antigen, low blood fibrinolytic activity are all risk factors for coronary disease and possibly for stoke.

HAEMATOCRIT

The association of stroke with haematocrit is confounded by cigarette smoking blood pressure and plasma fibrinogen.

ATRIAL FIBRILLATION

Most frequent potential cardiac source of embolism to the brain is atrial fibrillation by virtue of clot forming in left atrial appendages in developed countries the cause of atrial fibrillation is non rheumatic. The risk of first stroke is about five percent per year in non rheumatic atrial fibrillation. Lone atrial fibrillation (ie no other cardiac disease) seems still to be a risk factor unless so strictly defined than it represents less than ten percents of all fibrillation where embolic risk is minimal. People in atrial fibrillation with associated risk factors are at a higher risk of stroke compared to people with only AF. Most strokes associated with AF are in elderly.

HYPER COAGULABLE STATES

Antiohospholipid antibodies, lupus anti –coagulant, protein c, protein s, and factor V mutation.

SEX HORMONES

Stroke severity is associated with estrogen levels in women. Oral contraceptives triple the chances of stroke. About ten percent of stroke in women are due to oral contraceptives.

ALCOHOL

It is more of a risk factor for haemmorhagic stroke. It may be protective for ischemic stroke when taken in moderate amount.

OBESITY

Obesity as a risk factor in stroke mediated by hypertension and diabetes.

DIET

Exercise and moderate salt intake may be responsible for increasing the blood pressure. the reduced intake of vegetables vitamine- C betacarotene and flavinoids have all been proposed as vascular risk factors.

EXERCISE

Exercises reduces blood pressures, plasma cholesterols and fibrinogen and the risk of non insulin dependent dib3etes mellitus. So perhaps not surprisingly lack of exercise is associated with heart disease and stroke.

INFECTION AND INFLAMMATION

Blood cells has been associated with vascular events, there is of an association between stroke and serum c-reactive protein and various immediate preceding and distant infection.

HOMOCYSTEINEMIA

Homozygous patients with a race inherited deficiency of cystatnosine synthase develop severe homocystienemia and homocystenuria and present as maenyal retardation, dislocated lenses marfanoid features and atendecies to arterial and venous thrombosis. H eterozygotes have modestly raised levels of homocystiens as do the much more common heterozygotes for methylenetetrahydrofolate deficiency and patients who are patients who are mildly defecients in the co-factors to these enzymes- folic acids, vitamins b12, pyridoxines. There is enough reasonably consistent observational dara linking stroke and coronary disease with increasing homocystiene for trials of folic acids and pyridoxine have been started.

GENETIC FACTORS

Few strokes are familial parental history of strokes is a risk factor. Factors like hypertension and hyperlipidemia which contribute to strokes are genetic. Cerebral Autosomal Dominant Arteriopathy with Sub-cortiol Infarcts and Leukoencephalopathy is a rare syndrome in whih migrane with aura develops in the 30s, reccurant lacunar ischemic strokes and TIAS start

in the 40s sub cortical dementia develops in the 50sand patients die in the 60s. Genetic locus is on chromosome 19 q12.

Anatomical Variation

Anatomical variation in cerebral vessels by itself is a risk factor for stroke occurrence.

CLINICAL FEATURES

Patients usually present with a wide variety of symptoms of neurological dysfunction includes hemi or quadripareis, cranial nerve defects (III – XII), respiratory difficulty, altered sensorium, vertigo and ataxia. Multiple cranial nerve signs indicate involvement of more than one brain stem level. Patients may present with only hemi paresis, which may progress rapidly to quadriparesis or a locked in syndrome. The onset of symptoms may not be as abrupt as with anterior circulation strokes[27].

As the posterior circulation supplies the brainstem, cerebellum, and occipital cortex, the symptoms frequently involve were dizziness, diplopia, dysarthria, dysphasia and dyslexia[28].

The hallmark of posterior circulation stroke is crossed findings, with cranial nerve finding on the side of the lesion and motor sensory findings on the opposite side. The exact symptom depends on the precise location of infarct[29.]

VERTEBRAL ARTERY SYNDROME

Occlusion of one vertebral artery is usually asymptomatic. This is due to adequate collateral blood from the opposite vertebral artery or extracranial vessels.

When both the arteries are atretic, the blood flow is by the basilar artery through the retrograde flow by the posterior communicating artery and by the extra cranial vessels. Brain stem ischemic stroke may occur.

Subclavian steal syndrome, in stenosis of the subclavian artery proximal to the origin of the vertebral artery. on exerting the arm there may be retrograde flow down the vertebral artery. Mostly it is asymptomatic and rarely can result in vertebral insufficiency. Surgical correction or by pass of the subclavian artery may be needed.

Lateral Medullary Syndrome – in the absence of collateral blood flow, the occlusion of vertebral artery causes infarction of wedge shaped area of the lateral medulla and the inferior cerebellum called lateral medulla also called WALLENBERG SYNDROME[30]. Less often in 30% of cases, this syndrome is resulting from posterior inferior cerebellar artery (pica) occlusion.

The characteristic clinical picture consists of

a) Damage to the nucleus ambiguous or the issuing fibres of IX, X nerves traversing the medulla result in ipsilateral paralysis of palate, pharynx

and larynx resulting in nasal regurgitation, nasal voice, dysphagia and dysphonia.

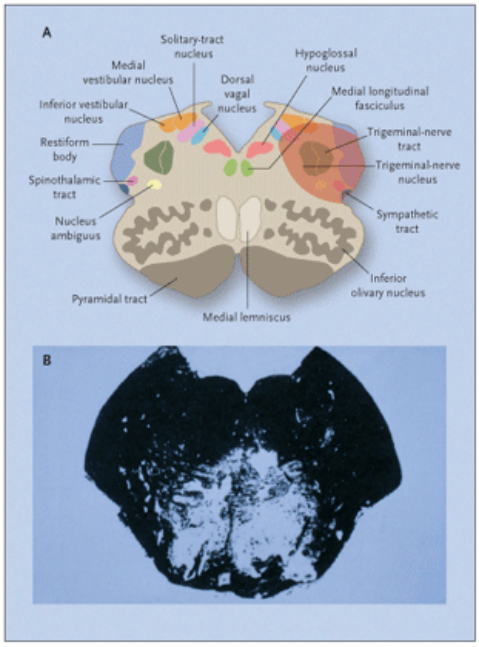
b) Involvement of the spinal tract and nucleus of the V nerve results in ipsilateral loss of pain and temperature sensation over the face.

c) Infarction of the lateral spinothalamic tract causes loss of pain and temperature sensation over opposite half of the body

d) Infarction of descending sympathetic fibres in the medullary reticular formation usually produces a partial rather than a complete horners syndrome on the same side.

e) Infarction of the inferior cerebellar peduncle and or cerebellum sesulting in nestagmus, which is maximal on looking to the side of the lesion and ipsilateral ataxia.

f) Damage to the vestibular nuclei causes vertigo associated with nausea vomiting and nystagmus.



MEDIAL MEDULLARY SYNDROME:

Atheromatous occlusion of the medullary penetrating branches of the vertebral artery or posterior inferior cerebellar artery result in infarction of the central portion of the medulla adjacent to the midline.

Damage to the pyramid is causing a contralateral hemiparesis of the arm and leg sparing the face.

Infarction of the medial leminiscus and the hypoglossal fascicle is producing contraletral loss of position and joint sense and ipsilateral tongue weakness.

BASILAR ARTERY SYNDROME

Paramedian Branch Occlusion

1. Thrombosis of small PENETRATING arteries entering the caudal pons produces paramedian infarction resulting in
   a) contralateral hemiplegia due to the involvement of the corticospinal tract
   b) ipsilateral lateral rectus paralysis (VI cranial nerve) with diplopia that is accentuated when the patient looks towards the lesion.
   c) Ipsilateral facial weakness as the vii nerve fibres are interrupted as they sweep round the vi nerve nucleus.
   d) Conjugate gaze palsy to the side of the lesion due to damage to medial longitudinal fasciculus.

e) Contralateral loss of light touch, proprioception due to the damage to medial leminiscus.
f) Ipsilateral cerebellar ataxia due to the involvement of potine nuclei and the fibres passing to the middle cerebellar peduncle.

A, b, and c are collectively called as millard-gubler syndrome. A, c and d are together referred as foville syndrome.

2. BILATERAL LESIONS OF BASIS PONTIS; bilateral occlusion of penetrating vessels supplying the ventral pons results in locked in syndrome, which consists of; quadriplegia due to bilateral corticospinal involvement.

The patient is mte due to the involvement of bilateral corticobulbar fibres destined for lower cranial nerves.

Impairment of horizontal eye movements as the vi nerve fascicles are damaged. Blinking and vertical eye movements are intact since the supranuclear oculomotor pathways and facial nerve fascicles are spared.

The patient is fully conscious as the reticular formation is not involved.

Spinothalamic sensations are retained but the involvement of the medial leminiscus produces loss of proprioceptive sensation and touch over the entire body.

LONG CIRCUMFLEX BRANCH OCCLUSION

Superior cerebellar artery syndrome; is caused by the infarction of dorsolateral aspect of upper pons and midbrain and dorsal surface of the cerebellum. The characteristic features are;

Ipsilateral cerebellar ataxia due to the involvement of the middle and superior cerebellar peduncles and superior surface of cerebellum.

Horner's syndrome (miosis, ptosis, decreased sweating over face) due to the lesion of descending sympathetic tract.

Involvement of spinothalamic tract results in contralateral loss of pain and temperature sensations over the face and the body.

Loss of touch, proprioceptive sensations over the opposite leg due to the lesion of the lateral most portion of the medial leminiscus.

Anterior inferior cerebellar artery sundrome; is resultinf from infarction of lateral tegmentum of lower pons and ventrolateral surface of cerebellum.

Ipsilateral cerebellar ataxia due to the involvement of middle cerebellar peduncle and cerebellum.

Ipsilateral loss of pain and temperature over the face due to the damage to descending tract and nucleus of V nerve.

Ipsilateral facial weakness due to the lesion of VII nerve fascicle.

Conjugate gaze palsy to the side of the lesion resulting from involvement of the centre for conjugate lateral gaze(PPRF)

Deafness, nystagmus and vertigo associated with nausea and vomiting due to the lesion of vestibular and cochlear nuclei.

Horner's syndrome due to the damage to descending sympathetic pathway.

Loss of pain and temperature over the opposite half of the body resulting from the lesion of spinothalamic tract.

SHORT CIRCUMFERENTIAL ARTERY SYNDROME

Thrombosis of short circumferential branches result in infarction of the lateral tegmentum of the pons. The characteristic features are;

Ipsilateral cerebellar ataxia is due to the lesion of the middle cerebellar peduncle.

Ipsilateral loss of sensations over the face due to involvement of the sensory root or nucleus of the V cranial nerve.

Paralysis of muscles of mastication resulting from the lesion of the motor root or nucleus of V cranial nerve.

Loss of pain and temperature over the opposite half of the body due to the involvement of the spinothalamic tract.

Horner’s syndrome is usually present on the affected side.

Contralateral weakness of the leg due to associated lesion of the lateral portion of the corticospinal fibres.

COMPLETE OCCLUSION OF BASILAR ARTERY

Complete occlusion of main trunk of basilar artery is catastrophic with rapid onset of coma and is usually fatal. It leads to profound coma,

quadriplegia, pseudobulbar palsy and cortical blindness with small fixed pupils.

Survivor show signs of bilateral brainstem involvement with the recovery of consciousness. Corticospinal tract damage result in fquadriplegia with severe spasticity. Involvement of lateral spinothalamic tract and medial leminiscus result in bilateral sensory loss. Patients also show features of bilateral cranial nerve dysfunction like facial paralysis, impairment of ocular movements, loss of sensations over face and pseudobulbar palsy. This may be coupled with signs of occipital and temporal lobe infarction like permanent cortical blindness and amnesia.

OCCLUSION OF TERMINAL PORTION OF BASILAR ARTERY

(Top of basilar syndrome) also called Caplan's syndrome

Occurs when a embolus impacts at the termination of the basilar artery where both the posterior cerebral arteries arise. This has been refered as saddle embolis. Saddle embolism produces occlusion of both the posterior cerebral arteries and if extensive also produce occlusion of both superior cerebellar arteries. The characteristic features are;

Oculomotor disorders like impairment of vertical gaze and convergence, skew deviation of eyes and small poorly reacting pupils are due to infarction of pretectal area of midbrain.

Cortical blindness due to the infarction of both the occipital lobes.

Anton's syndrome (deniel of blindness) due to extensive lesions of visual association areas.

Behavioural abnormalities and amnesia due to the bilateral lesions of medial aspect of temporal lobe.

POSTERIOR CEREBRAL ARTERY SYNDROME

The clinical syndrome depends upon the site of occlusion and availability of collateral circulation.

PRECOMMUNAL SYNDROME

Occlusion of penetrating branches to the midbrain results in ischaemia or infarction involving cerebral peduncle, third nerve nucleus of fascicle, the red nucleus and cerebellar connections.

SITE OF LESION	CLINICAL EFFECTS
Oculmotor nerve and cerebral peduncle	Weber's syndrome Ipsilateral third nerve palsy and Contralateral hemiplegia
Oculomotor nerve and red nucleus	Claude's syndrome Ipsilateral oculomotor paresis With contralateral ataxia & tremor.
Oculomotor nerve and brachium Conjunctivum	Nothangel syndrome ipsilateral oculomotor paresis with ipsilateral ataxia

Oculomotor nerve and subthalamic Nucleus	Benedikt's syndrome ipsilateral oculomotor paresis with contralateral ataxia and Hemichoreoathetosis.
Occlusion of penetrating branches to the subthalamic nucleus of Luys	Contralateral hemiballismus with involuntary flinging movements of the affected limbs & hemiataxia
Dorsal mid brain	Parinaud syndrome Characterized by paresis of upward gaze; defective convergence & divergence, light near dissociation of pupils (pupils are large, not reacting to light, but accommodation is spared); convergence retraction nystagmus; skew deviation of eyes.
Occlusion of the thalamogeniculate Branches produce infarction of the Ventral posteromedial nuclei of Thalamus.	Thalamic syndrome (Dejerine & Roussy's) contralateral sensory loss of Both superficial sensations (touch, pain, temperature) and deep sensations (proprioception).

After a few weeks or months there is development of parasthesia or severe excruciating dysesthesia (thalamic pain) in the affected areas. This thalamic pain poorly responds to analgesics, but anticonvulsants may be beneficial.

Mild transient hemiparesis may be associated due to the involvement of the posterior limb. This usually resolves with some residual spasticity and increased reflexes.

Involuntary movements (choreoathetosis, hemiballismus, intention tremor) may be present.

POSTCOMMUNAL SYNDROME

Occlusion of posterior cerebral artery distal to the posterior communicating artery cause infarction in the territory of cortical or hemispherical branches (ie. Infarction in the medial aspect of the occipital and temporal lobe.)

The clinical syndrome depends upon the site of occlusion and availability of collateral circulation.

SITE OF LESION	CLINICAL EFFECTS
Unilateral lesion of visual cortex	Elementary visual hallucinations; Contralateral homonymous hemianopia With a macular sparing since the macular region of the visual cortex is supplied by the middle cerebral artery.
With bilateral lesion	cortical blindness characterized by Visual loss in both eyes in the presence of normal pupillar reflexes and optic disc. Sometimes peripheral vision is lost and central vision is spared, resulting in gun-barrel vision.
Unilateral lesions of visual Association cortex. (lesions with respect to dominant hemisphere)	visual object agnosia visual verbal agnosia (alexia without agraphia) also called word blindness.
With bilateral lesions	Balint's syndrome Characterized by optic ataxia (inability to visually guide limb movements), ocular ataxia (inability to move eyes to a precise point in visual

	field), inability to enumerate objects in a picture, extract meaning from a picture and to avoid objects seen in one's path. Prosopagnosia (agnosia for familiar faces) Anton's syndrome (denial of blindness) visual agnosia for colours. Oculomotor apraxia. Visual agnosia for space.
Lesions of mesial aspect of temporal lobe (dominant hemisphere)	Acute memory disturbances, but the defect is transient as memory has bilateral representation.
Bilateral lesions	Amnestic syndrome Acute onset of agitated delirium.

Lacunar strokes:

Refers to infarction following atherothrombotic or lipohyalinotic occlusion of one of the small vessels. They range in size from 3-4 mm to 1-2 cm. Hypertension and age are the principal risk factors. Lacunar infarcts cause approximately 20% of all strokes.

Brainstem lacunar infarcts:

Produce a wide range of symptoms and signs

1. Pure motor hemiparesis from an infarct in the posterior limb of internal capsule or basis pontis.
2. Pure sensory stroke from an infarct in ventrolateral thalamus.
3. Ataxic hemiparesis from an infarct in the base of the pons.
4. Dysarthria and a clumsy hand or arm due to infarction in the base of the pons or in the genu of internal capsule.

Syndromes resulting from occlusions of the penetrating arteries of the basilar artery include ipsilateral ataxia and contralateral crural paresis, hemiparesis with horizontal gaze palsy, and hemiparesis with a crossed sixth nerve palsy. Lower basilar branch syndromes include internuclear opthalmoplegia, horizontal gaze palsy and appendicular cerebellar ataxia.

Vertebrobasilar insufficiency (VBI) is a term used to describe fluctuating brainstem symptoms, such as dizziness associated with cranial nerve symptoms or cerebellar dysfunction over a period of days to weeks. This indicates insufficient flow through the posterior circulation and is essentially a brainstem TIA. Rarely VBI present as vertigo alone [31].

CURRENT MANAGEMENT OF ACUTE STROKE

Actually stroke management dose not concern only acute treatment but also patient management. The lack of efficacy of acute treatments may be related to the fact that many patients are referred and treated too late. Ideal managements of stroke patients implies a rapid evaluation of the neurological condition, the performance of selected investigations, and the

managements of the patients in a specific stroke care unit. We must emphasize the importance of clinical evaluation. Which is the basis for the choice and timing of investigations as well as for evaluating the efficacy of treatment[32].

Laboratory investigations in acute stroke

The choice and the timing of laboratory investigations are fundamental. However it is difficulty to establish a standard protocol of testing because the evaluation of patients depends on patient characteristics, stroke topography, and the possible existence of others medical problems. Laboratory investigations in acute stroke include two steps first the confirmation of stroke, and second the determination of the most likely etiology of stroke.

Investigations to confirm the diagnosis of stroke

Brain computed tomography:-

Computed tomography (CT) is the key for diagnosis and the pursuit of future investigations and is usually necessary before treatments. CT scanning on an emergency basis is now available at most medical centers. It is safe, non invasive and commonly done without contrast administrations, which readily allows the clinical to exclude intracranial hemorrhage and space – occupying lesions such as tumor or abscess. When CT scanning is performed within the first 6 hours after stroke onset and ischemic lesion is not usually visulised, but in this particular situation, indirect infarct signs such as a spontaneous hyperdense middle artery or vanishing of the cortical – subcortical gradient can be useful indications of large infarctions leading

to a poor prognosis. However CT scanning has other limitations: brain stem and posterior inferior cerebellar artery territory infarcts are not often seen even later after stroke onset, and small cortical or subcortical infarcts can sometimes only be seen after contrast enhancement. When baselines CT scanning is normal, a second study 1 week later is commonly recommended.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is the best examination for the diagnosis and localization of ischemic stroke. Compared to CT scanning, MRI is more sensitive in detecting recent stroke. Actually the high sensitivity of MRI allows detection of early signs of infarct a few hours after stroke, mainly with a decrease in the cortical – subcordical gradient.

Because there is less bone artifact, MRI is the first choice for detecting brain stem or cerebellar infarcts. MRI scan improve stroke localization and detect small infarcts. The use of gadolinium - meglumine gadoterate (DTPA) for contrast enhancement can define the age of an ischemic lesion. Vascular occlusion or low flow can be suggested on MRI, especially in large vessels, and thrombus and embolus can sometimes be distinguished. MRI also allows early detection of hemorrhagic transformation of ischemic stroke. The disadvantages of MRI are difficulties in ultra early diagnosis of hemorrhage the cost of the technique. The long acquisition time, necessitating full cooperation of the patient, which is not often available in stroke and its specificity, which may lead to detection of minor changes in brain water content without any clinical correlations.

Investigations of determine the etiology of stroke

Because the differential diagnosis of the cause of ischemic stroke is broad and because the complete evaluation is potentially extensive and expensive the rationale for etiologic investigations must be tailored to the individual patients. We must perform an exhaustive search for less common causes of stroke mainly for the following groups. Children and adults under the age of 45 and patients without obvious risk factors for stroke. However in some cases, despite extensive investigations the etiology of stroke remains unknown.

Hematologic investigations

Hematologic investigations are usually the first acknowledged and obtained investigations and may be performed in the emergency room. These first tests generally include an erythrocyte sedimentation rate, red and white blood cell counts, platelets count, serum enzymes, cholesterol and lipids, and routine coagulation profile. Careful attention must be given to the coagulations studies. Actually in most cases a routine coagulation profile, including serum fibrinogen, prothrombin time, and partial thromboplastin time is sufficient. However, in stroke patients without risk factors for stroke a coagulation disorder with a congenital defect in protein C or S or antithrombin III can be present. In other studies, antiphospholipid antibodies were suspected to be the cause of stroke. The tires of these proteins or antibodies must be obtained in the absence of either platelets antiaggregant or anticoagulant therapy to be reliable. We suggest that these analyses should be performed as soon as possible in selected patients.

However the significance of abnormal findings has yet to be clarified as the ischemic event or associated conditions can themselves induced congulation changes. We recommend interpreting these results cautiously and in case of doubt reconfirming them several weeks after stroke.

Noninvasive studies

Noninvasive studies include echocardiography to look at the heart and noninvasive vascular studies of the cervical and intracranial arteries. In suspected cardioembolism, the investigation must include at least 24 hours of three – lead electrocardiography (ECG) monitoring and, in selected patients, exercise ECG.

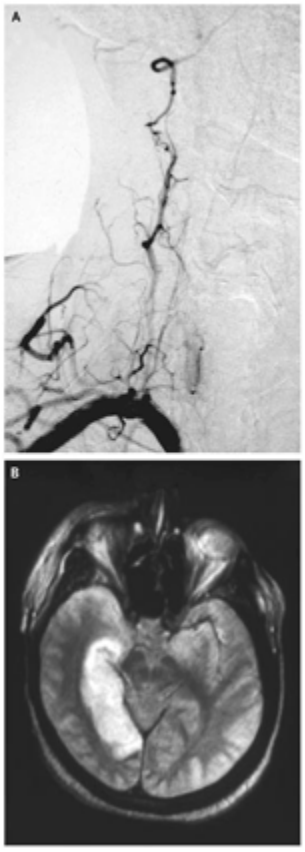
Echocardiography – the role of ECG is crucial among investigations of stroke patients, because cardioembolism is the cause of roughly 25-30% of ischemic stroke. The neurologic presentation is often suggestive of cardioembolism. Transthoracic two – dimensional ECG is now widely available. It gives reliable information on the ventricular wall as well as the aortic and mitral valves. It can exclude a left ventricular thrombus. When performed with contrast it can demonstrate intracardiac shunts, which are frequent in young adults with stoke. However the clinical significance of these abnormalities remains controversial in some situations. the disadvantages of this technique are that some cardiac abnormalities are less well demonstrated than with transophageal ECG. Moreover studies in obese patients are not reliable.

Transesophageal echocardiography – Transephageal ECG represents an important advance for stroke patients. It is critical for the investigations of the posterior part of the heart, particularly the left atrium and appendage. It can also provide information on atheromatosis and ulcerated plaques located in the aortic arch. Since it is semi- invasive, it must be considered only in patients highly suspect for cardioembolism, in whom transthoracic echocardiography is normal, or unreliable. The disadvantages of this technique are essentially due to the endoscopic nature of the procedure, which necessitates good cooperation between the and the physician. Rarely electrophysiologic studies or exercise electrocardiography may contribute to the determination of stroke etiology.

Duplex investigations :- Duplex imaging of the extracranial carotid and vertebral arteries is easy, and noninvasive. It can provide information on a possible arterial source of emboli and on arterial occlusion. Duplex imaging is currently performed as a screening procedure before more invasive investigations, such as cerebral angiography. Unfortunately, it will probably not replace cerebral angiography prior to carotid surgery, because it does not fully explore the carotid siphon and cerebral vessels. It can be combined with transcranial Doppler, which provides information on the cerebral circulation. Recently its role in the detection of symptomatic cerebral emboli has been emphasized.

Electroencephalography- electroencephalography (EEG) does not have an essential role in acute stroke, but it can provide information about stroke localization (deep versue superficial) and sometimes it allows the clinical to differentiate migraine from stroke.

Magnetic Resonance Angiography :- Magnetic resonance angiography (MRA) gives information on blood vessels and blood flow. It may one day replace conventional arteriography. Use of MRA has the advantage of being noninvasive and gives a technical resolution comparable to that of conventional angiography, with the possibility fro three dimensional images. However this technique is only available in some academic centers, and it s const and poor availability usually limit its use to selected patients.



Conventional angiography:- Conventional angiography is used to determine the presence of abnormalities in both intra – and extracranial vessels. In the first hours following stroke, small cerebral artery occlusions can be demonstrated, but in the management of acute stroke, its role remains

controversial. In central nervous system vasculitis, abnormalities of cerebral vessels can be demonstrated on angiography, but the sensitivity in only about 50%.

Complications of this technique are not rate, though permanence complications are less than 0. 55 in frequency. Presently angiography is performed acutely only in selected cases.

Cerebrospinal fluid investigations:- Cerebrospinal fluid (CSF) examination is rarely required for a patients with acute ischemic stroke. It can provide information in cases of cerebral venous thrombosis by detecting red blood cells and high CSF pressure, as well as in cases of vasculitis.

Other Investigations – other investigations are rarely performed in acute stroke, skin biopsy can be done when a cutaneovascular syndrome, vasculitis, or collagen abnormalities are suspected. Urine tests can also be performed in young patients in the absence of well – known stroke risk factors to exclude hetrozyous forms of homocystinuria or to exclude the presence f drugs such a cocaine, amphetamine like substances or cannabinoid in addicted patients.

MATERIALS AND METHODS

This study was carried out in patients who presented with posterior circulation ischemic stroke to the medicine and neurology department of PSG Institute of Medical Sciences and Research, Coimbatore.

The study was conducted during the period of January 2005 to march 2006.

All patients admitted with clinical features suggestive of posterior circulation stroke were taken. All were subjected to CT scan brain. Patients with evidence of posterior circulation stroke clinically and imaging wise were taken up for the study.

INCLUSION CRITERIA

All patients with clinical features suggestive of posterior circulation stroke Imaging showing infarcts within the posterior circulation territory.

EXCLUSION CRITERIA

CT evidence of showing haemorrage

Patients having evidence of infarcts in other areas ie. , territory of anterior circulation, border zone infarcts.

Patients on ventilator, global aphasia, in coma were excluded from studies.

All the patients were evaluated in detail. A detail history was recorded with special emphasis on risk factor, mode of presentation, time interval between onset and arrival to hospital.

Major risk factor included in this study are hypertension, diabetes mellitus, dyslipidemia, age sex, coronary artery disease, smoking, alcohol intake, and transient ischemic attack

Special emphasis was given for clinical evaluation of cardiovascular system with regard to rhythm disturbances cardiac failure, and valvular heart disease.

Detailed clinical neurological examination was done and the patients were grouped into posterior circulation stroke

Blood haemogram, peripheral smear, haematocrit, blood sugar lipid profile, and renal function tests were done

Echocardiogram was done. to rule out intracavitary clots and vegetation. Imaging studies including CT, MRI and Doppler Studies were done to localize the anatomical area involved and to know the size of the area involved.

Outcome was assesed according to the modified rankin scale at 30 days.

PROFORMA

NAME

AGE

SEX ADDRESS

IP NOS

DOA DOD

DETAILED CLINICAL HISTORY AND EXAMINATION with emphasis on the following like giddiness, weakeness, slurring of speech, cerebellar signs, sensorium, loss of conciousness, seizure, headache visual disturbances, time interval and risk factors was done.

GENERAL EXAMINATION

PULSE

BP

EXAMINATION OF CENTRAL NERVOUS SYSTEM

EXAMINATION OF OTHER SYSTEMS

INVESTIGATIONS

CBC

DIABETIC PROFILE

LIPID PROFILE

RENAL FUNCTION TESTS

ECG

CHEST X-RAYS

ECHO

CT

MRI

CAROTID AND VERTEBRAL DOPPLER

MODIFIED RANKING SCALE

0 no symptoms

1. No significant disability able to carryout daily activities
2. Slight disability to carryout daily activities with assistance
3. Moderate disability. Needs help. Able to walk.
4. Moderately severe disability. Needs assistance for ADL and walk.
5. Severe disability. Bed ridden, incontinent, nursing care required.
6. Dead

OBSERVATION

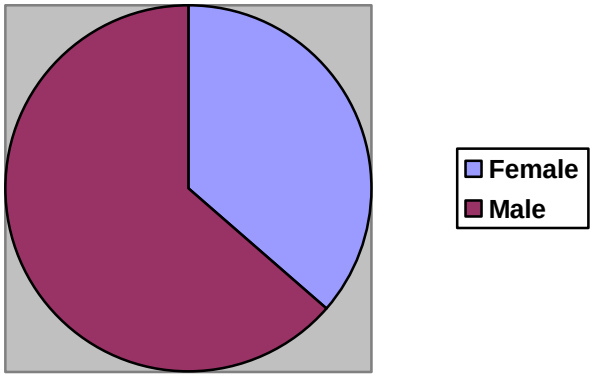
80 patients admitted with clinical features suggestive of posterior circulation stroke were enrolled in the study.

TABLE NO: 1

SEX INCIDENCE OF POSTERIOR CIRCULATION ISCHEMIC STROKE IN 80 CASES

Among 80 patients 51 patients were males (63. 75%) and 29 females(36. 25%).

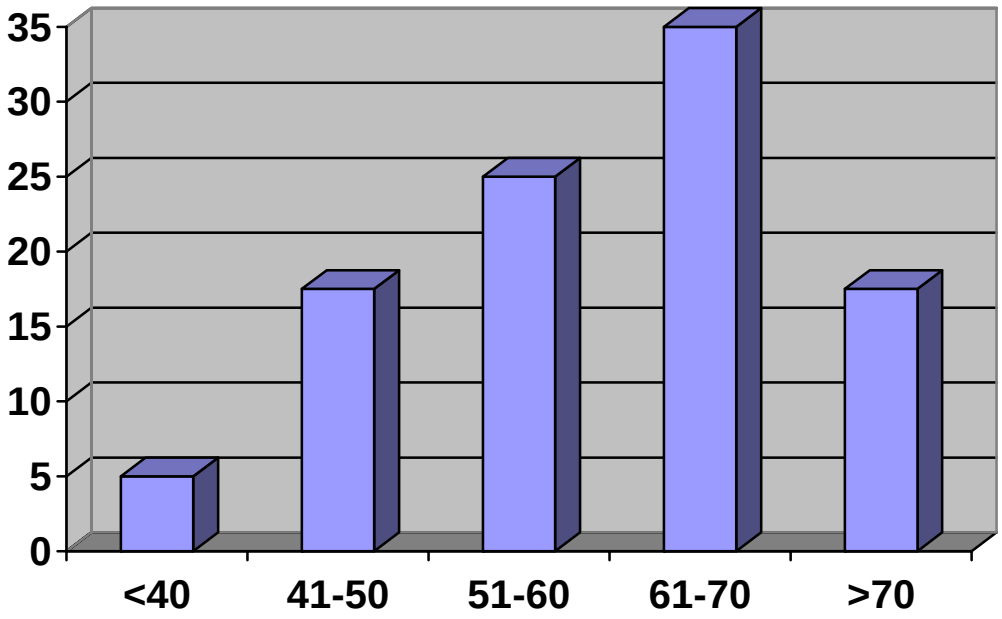
SEX	NO OF CASES	PERCENTAGE
MALE	51	63. 75
FEMALE	29	36. 25
TOTAL	80	100



AGE INCIDENCE:

Age group distribution is shown in table 2. The age group ranges from 40 to 70 years. Maximum numbers of cases were in the age group 60 to 70.

AGE IN YEARS	< 40	41-50	5-60	61-70	ABOVE 70
NO OF CASES	4	14	20	28	14
PERCENTAGE	5	17. 5	25	35	17. 5



RISK FACTORS:

The possible risk factors were studied in all 80 patients. 42 patients had hypertension and 30 patients had diabetes mellitus. 25 patients gave h/o smoking. The risk factors identified were shown in table 3.

Table 3.

RISK FACTORS	NO OF CASES	PERCENTAGE
HYPERTENSION	42	52. 5
DIABETES	30	37. 5
DYSLIPIDEMIA	16	20
IHD	17	21. 25
SMOKING	25	31. 25
ALCOHOL	12	15
TIA	8	10
CARDIOEMBOLIC	8	10
HAEMATOCRIT	18	22. 5
UNCERTAIN	14	11. 2

CLINICAL FEATURES

The clinical features at the onset of stroke were studied. Most of our patients presented with giddiness and vomiting. Headache was present in 22 patients. 14 patients had altered sensorium at onset. 8 patients had seizures at the onset (table 4).

The other clinical features that were present in this study were according to the territory involved. this included homonymous, heminanopia, temporal lobe sign, cerebellar signs, weakness, sensory disturbances, cranial nerve disturbances (III, IV, V, VII, IX and X) most of these were combination of clinical (table 5).

Table 4.

S. No	Clinical features	No of pts.
1.	Giddiness and vomiting	52
2.	Headache	22
3.	Seizures	8
4.	Altered sensorium	14

Table 5.

Clinical features	No of pts.
Visual field defect	20 (25%)
Cerebellar signs	30 (37. 5%)
Weakness	38 (47. 5%)
Hemi sensory loss	12 (15%)
Cranial nerve involvement	28 (35%)
Combination	45 (55%)

Anatomical areas involved

To describe the location of infarcts, we subdivided the posterior circulation in to proximal, middle, and distal intra cranial arteries accordingly described by NEMC posterior circulation registry. The clinical features and neuroimaging are taken together to describe the location of infarct. For about 12 patients, in whom we had a normal CT and MRI was not done due to financial constrains, only the clinical features were taken to locate the territory of infarcts.

Among these 80 patients we found that distal territory involvement was more common. Isolated middle and proximal territory infarcts were less in this study. In other patients we had varying combination of either proximal middle or distal territory infarcts. The location of terrirory was shown in

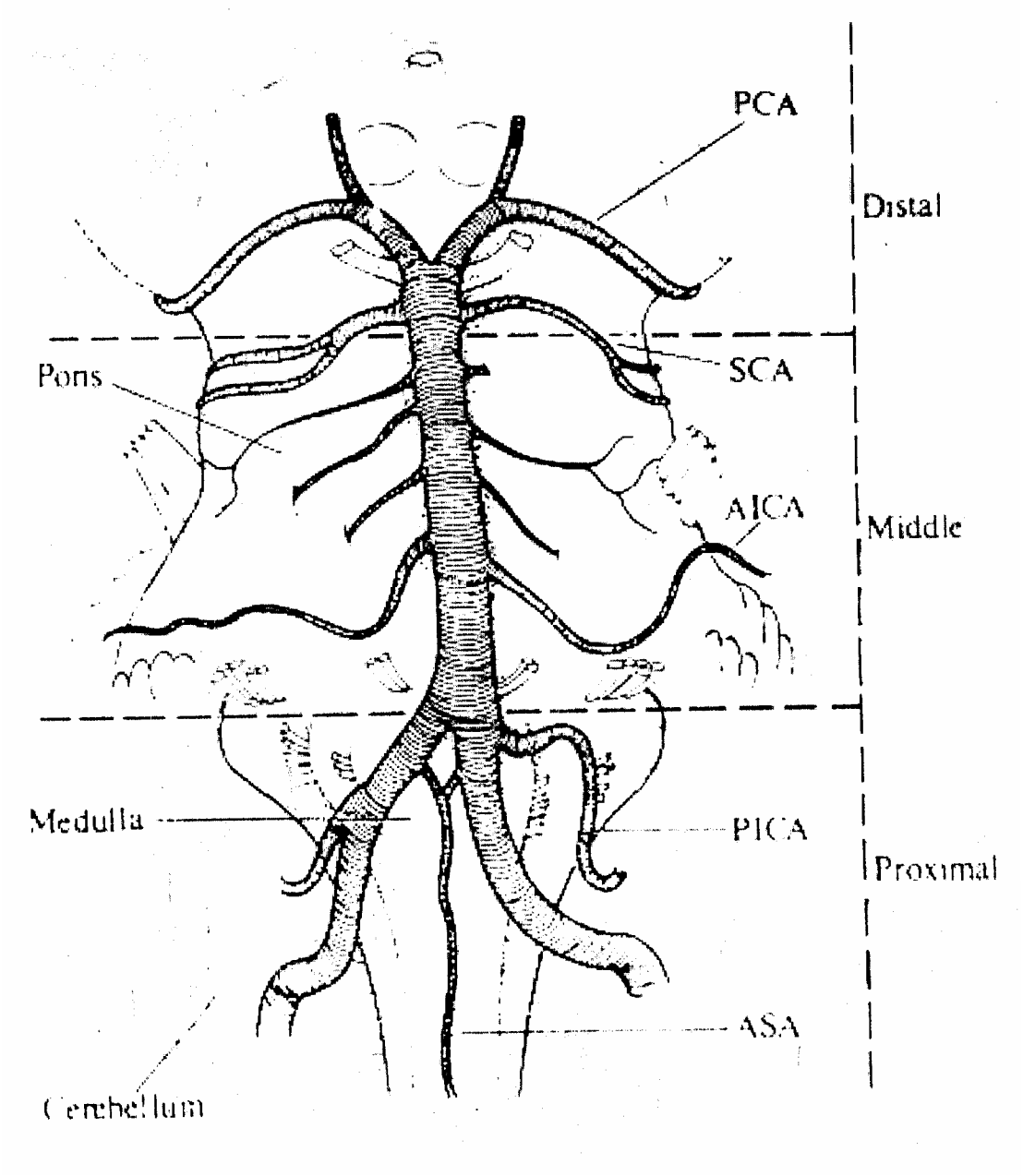


Table 6.

Location of infarctions	**No of pts**
1. distal only	36 (40%)
2. proximal only	9 (11. 25%)
3. middle only	12 (15%)
4. proximal and middle	3(3. 75%)
5. proximal and distal	11 (13. 75%)
6. middle and distal	12 (15%)
7. proximal, mid, and distal	8(10%)

Table 7.

AREAS INVOLVED	NO OF CASES	PERCENTAGE
CEREBELLUM	29	36. 25
BRAIN STEM	32	40
THALAMUS, MEDIAL TEMPORAL, OCCIPITAL	19	23. 75

SIZE OF INFARCT :

SIZE OF THE INFARCT	NO OF CASES	PERCENTAGE
LARGE VESSEL	58	72. 5
LACUNAR	16	20
CARDIOEMBOLIC	6	7. 5

OUTCOME AT 30 DAYS :

STATUS OF PATIENTS	FREQUENCY
NO DISABILITY	30
MINOR DISABILITY	48. 3
MAJOR DISABILITY	16. 3
DEATH DUE TO CEREBROVASCULAR CAUSES	2. 5
DEATH DUE TO OTHER CAUSES	1. 9

DISCUSSION

Cerebrovascular disease was more common in men in the age group of >60 years. In 'tufts posterior registry' 52% were male and 48% were females. Smajlonc D[33] studied ischemic insult in both anterior and posterior circulations and he found 18. 2% had posterior circulation stroke and he also found females and males were equally affected in posterior circulation stroke.

In this study 63. 75% were males. Though the above mentioned study showed sexual equality our study showed a male preponderance.

Mean age of pts in our study was 60 years which was similar to other studies.

The risk factors in stroke are classified as modifiable and non modifiable. Male sex and older age are non-modifiable risk factors.

Hypertension is the most common risk factor associated with stroke. In our study 52. 5% were hypertensives this was similar to the stoke study of Nizam's Institute Hyderabad[34].

Diabetes mellitus, hypercholesterimia, smoking, obesity, ischemic heart disease, atrial fibrillation, heamatocrit elevation are other modifiable risk factors.

Smoking was found in 31. 25%. Diabetes 37. 5%, dispidimia 20%, cardio embolic strokes 10%. Huan et al[35] found that 71% of his pts had

hypertension and 38% of pts had smoking history. Comparison of risk

Risk factors	Present study	Huan et al
Hypertension	52. 5%	71%
Diabetes	37. 5%	22. 6%
Smoking	31. 5%	38. 7%
Ischemic heart disease	21. 25%	19. 4%

factors between their study and our study is shown in table 8.

Table 8:

Clinical features at the onset of stroke in our study were giddiness and vomiting. The other presentation in our study in our study included headache, altered sensorium, and seizures.

Timothy et al had described vertigo without hearing loss as the commonest symptom in brain stem stroke syndromes. Vertigo occurs both in small vessel and large vessel disease. Huans et al had found 30% of his pts had vertigo

In this study, vertiginous onset was present in 60% patients with distal territory infarct and also in all patients with two or more territories involvement. of the persons with multiple territory involvement if middle territory involvement is also there, vertiginous onset was more common. Only in 25% of patients with isolated proximal territory infarct vertigo at onset was present. We found that all patients who had middle territory infarcts (pons and AICA supplied cerebellum) had vertigo onset.

The vertigo in middle territory infarct could be explained by involvement of vestibular nucleus and its connections in the pontine region.

Altered sensorium at the onset is seen in 17. 5% of patients in this study. HUANS ET AL had found only 6% had altered sensorium.

In this study among the 14 patients, with altered sensorium at the onset, distal territory was involved in 8 out of 14 patients, middle territory involved in 7 patients. Patients who had proximal territory infarcts did not have altered sensoruim.

In this study 47% had pyramidal and 36. 2% had cerebellar signs. Visual field defects were seen in 25%, 35% had cranial nerve involvement. 15% had hemi sensory loss and 20% had temporal lobe signs. HUAN ET AL study had described pyramidal signs in 58% and cerebellar signs in 51%.

CT brain was normal in 25 pts in our study. MRI brain was done in 50 pts. Among them 2 pts who had clinical features suggestive of lateral medullary syndrome had normal study of CT and MRI brain.

Regarding the vascular territory involvement in this sudy we have found that 40% had isolated distal territory involvement. The other territory involvement and multiple territory involvement was less.

In New England Medical Center posterior circulation registry[36], they found that distal territory involvement was more common.

Table 9:

Teritory	Present study	NEMC registry
Distal only	40%	40. 9%
Proximal only	11. 25%	18. 2%
Middle only	12%	16. 1%
Proximal and middle	3%	3. 45%
Proximal and distal	11%	8. 93%
All	8%	2. 5%
Middle and distal	12%	9. 79%

SITE OF LESION

Cerebellum is involved in 36. 2% of patients. Brain stem involvement seen in40% and in 23. 75% of patients area involved included are thalamus, medial temporal and occipital. Combined lesions involving more than one area is seen in around 19% of patients. This study correlates with the study of NEMC Posterior circulatory stroke registry.

SIZE OF INFARCT

72. 5% of patients had large vessel disease. 20% had lacunar infarct, cardioembolic stroke is seen in 7. 5% of patients.

OUTCOME AT 30 DAYS

No disability 30%, minor disability 48. 3%, major disability 16. 3%, death due to cerebrovascular disease 2. 5%, death due to other causes 1. 9% is seen in this study which correlates with the NEMC Posterior circulation stroke registry.

SUMMARY AND CONCLUSION

In my study I found that

- Males were affected more than females
- Age group commonly involved was above sixty yrs.
- Hypertension, diabetes and smoking contributed the major risk factors followed by cardio embolic source
- Giddiness was commenest symptom at the onset
- Most of the patients had clinical signs of cerebellum and brainstem lesions.
- The territory commonly involved was mid-brain, cerebellum, thalamus, occipital and temporal lobes.
- MRI was found to be more sensitive than CT in identifying brain stem lesions
- Outcome in posterior circulation stroke was found to be better than anterior circulation stroke.

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MASTER CHART

S. No	Name	Age	Sex	HT	DM	Dys. Lip.	HCT	Smoking	ECHO	CT Brain	MRI
1	THANGAMUTHU	70	M	Y	Y	Y				Occipital Temporal Cerebral	Occipital Temporal Cerebral
2	KRISHNAVENI	45	F							Occipital	Occipital
3	KANNAMAL	60	F	Y	Y	Y				Occipital Temporal Cerebral	Occipital Temporal Cerebral
4	GOVINRAJ	52	M				Y	Y		Cerebellum	Cerebellum
5	NAVANETHAN	82	M					Y		Cerebellum	Cerebellum
6	NACHIMUTHU	60	M	Y	Y	Y				Occipital	Occipital
7	KULANTHAIRAJ	83	M	Y		Y				BS	BS
8	MARIMUTHU	40	M					Y		Cerebellum	Cerebellum
9	ARUMUGAM	75	M	Y		Y	Y	Y		Occipital Temporal Cerebral	Occipital Temporal Cerebral
10	SAVITHRI	85	F								MB
11	SUBRAMINAN	57	M	Y	Y				LV Clot/BVD	Occipital Temporal Cerebral	Occipital Temporal Cerebral
12	SHANTHI	34	F								Cerebellum
13	NAJAPPAN	70	M			Y				Cerebellum	Cerebellum
14	SAMIAPPAN	53	M				Y	Y		Cerebellum	Cerebellum
15	NACA\HAMMAL	85	F								Left Pons
16	PERIYANAYAKI	59	F	Y	Y	Y			LVH, DD	Occipital Temporal Cerebral	Occipital Temporal Cerebral
17	RAJU	51	M	Y						N	
18	BADHRINATH	46	M					Y		Occipital	Occipital
19	PONNUSWAMY	70	M	Y	Y					N	
20	VERAPATHRAN	53	M	Y	Y	Y	Y	Y	LV Clot/BVD	Occipital Temporal Cerebral	Occipital Temporal Cerebral
21	MOHAN	62	M		Y		Y	Y		N	
22	SHRINIVASAN	55	M		Y	Y		Y	LV Clot	Cerebellum	Cerebellum
23	PERUMAL	69	M	Y	Y		Y	Y	SD	Cerebellum	Cerebellum
24	VELLUMANI	43	M							BS	BS
25	MUTHUMANIKAM	62	M	Y						N	
26	KRISHNAN	67	M	Y				Y		Occipital Temporal Cerebral	Occipital Temporal Cerebral
27	SABAPATHY	55	M		Y	Y				Cerebellum	Cerebellum
28	KARUPANNA	75	M				Y	Y		Occipital Temporal Cerebral	Occipital Temporal Cerebral
29	SUNDRAMAL	57	F	Y	Y				LVD	Occipital	Occipital
30	SARSTHVTHI	85	F	Y						N	
31	AMMANI	75	F		Y					BS	BS
32	MEENASHI	67	F							Cerebellum	Cerebellum
33	VEELAUTHUM	55	M	Y	Y		Y	Y	LVD	Thalamic - right	Thalamic - right
34	SUBRAMINAN	64	M		Y					N	
35	MENGA	53	F	Y	Y				BVD	BS	BS

36	RAMASWAMY	85	M							Thalamic - right	Thalamic - right
37	MUTHUSWAMY	77	M		Y					Occipital	Occipital
38	CHENNIAPPAN	75	M	Y							ThalamUS
39	LAKSHMI	75	F	Y	Y				BVD	MB	MB
40	SUBBIAN	55	M								Pons - right
41	RANI	42	F							BS	BS
42	RADHAMMAL	59	F	Y						N	
43	PALANISWAMY	44	M								Pons
44	MYLATHAL	65	F	Y						N	
45	THANGARAJ	70	M	Y	Y	Y				Occipital Temporal Cerebral	Occipital Temporal Cerebral
46	CHANDRASEKAR	52	M	Y	Y		Y	Y		BS	BS
47	EASWARI	46	F							Occipital Temporal Cerebral	Cerebellum
48	SYRYIAN	67	M	Y	Y	Y	Y	Y		Cerebellum	Cerebellum
49	ABDUL RASAK	60	M	Y						OCC, Pons, MB	OCC, Pons, MB
50	PAPYAMMAL	69	F	Y						N	
51	ANGAPPAN	65	M	Y	Y	Y			BVD	Occipital Temporal Cerebral	Occipital Temporal Cerebral
52	CHINNAMAL	68	F	Y	Y				BVD	Cerebellum	Cerebellum
53	KARUPANNA	50	M				Y	Y		Occipital Temporal Cerebral	Occipital Temporal Cerebral
54	GEORGE	55	M				Y	Y		BS	BS
55	DHAMODRAN	63	M					Y		BS	BS
56	PALANISWAMY	70	M		Y	Y				Occipital Temporal Cerebral	Occipital Temporal Cerebral
57	SWAMINATHAN	49	M	Y							Lateral Medullary
58	KRISHNASWAMY	64	M	Y			Y	Y		BS	BS
59	ARAVIND	37	M				Y	Y		Pons	Pons
60	BADHURNEESA	45	F							MB	MB
61	VISALAKSHI	61	M	Y						N	
62	HARIHARAN	70	M	Y						BS	BS
63	SATHAKUMAR	62	M	Y						N	
64	PANCHALINGAM	46	M	Y				Y		BS	BS
65	KRISHNA \MOORTHY	65	M	Y	Y	Y	Y	Y	SD	Occipital Temporal Cerebral	Occipital Temporal Cerebral
66	BEGAM BEEVI	48	F		Y						B/L Infacts pons
67	PERIYAKAL	67	M	Y						N	
68	STELLA	70	F							Occipital	Occipital
69	SUMATHI	63	F	Y						BS	BS
70	CHANDRAN	44	M				Y	Y			B/L occipito thalamus
71	CHINNAN	65	M		Y						MB
72	RAMAN	70	M				Y	Y			Lt Lat medullay
73	KAVERI	63	F	Y	Y	Y			LV Clot	Occipital Temporal Cerebral	Occipital Temporal Cerebral

74	SELVI	42	F							Cerebellum	Cerebellum
75	RAVI	45	M							Occipital	Occipital
76	KUMAR	43	M					Y	Y	BS	BS
77	CHINNASWAMY	59	M		Y					BS	BS
78	RUKMANI	60	F			Y				Occipital Temporal Cerebral	Occipital Temporal Cerebral
79	RANGASWAMY	58	M							Cerebellum	Cerebellum
80	RAMAEE	59	F	Y						B/L cerebellum	B/L cerebellum